

Neuroborreliosis with extrapyramidal symptoms: a case report

Grażyna Biesiada, Jacek Czapiel, Iwona Sobczyk-Krupiarz, Aleksander Garlicki, Tomasz Mach

Department of Infectious Diseases, Division of Gastroenterology, Hepatology, and Infectious Diseases, Jagiellonian University School of Medicine, Kraków, Poland

Abstract: The disease of Lyme is a tick-borne infection. It involves skin, the nervous system, joints and the heart. *Spirochaeta Borrelia burgdorferi* is the etiologic agent of the disease. In the majority of cases, clinical symptoms, like migrating erythema, occur from 3 to 30 days, sometimes to 3 months after a bite from a tick. The early disseminated infection involves multiple migrating erythema, neuroborreliosis, arthritis, myocarditis and other organ-related symptoms. The late stage of chronic infection involves chronic atrophic leg dermatitis, neurological and rheumatological symptoms, and other organ-related symptoms which persist for above 12 months. The diagnosis of the disease of Lyme is based upon specific clinical symptoms confirmed by serologic tests. The two-step diagnostic protocol including the ELISA method, confirmed by the Western-blot test, is optimal. The present article describes a case of a 59-year-old man, a computer specialist, who often spends his free time walking in woods for recreation, and who was bitten by a tick 3 years before hospitalization. The bite resulted in migrating erythema that subsided without antimicrobial treatment. In spite of this, the man had not changed his hobby exposing himself to bites from ticks. One year later, multiple migrating erythema and extrapyramidal symptoms appeared without any other organ malfunctions. In the current year, the patient was admitted to the Infectious Diseases Hospital, and received antibiotics (ceftriaxon) with following neurological improvement. Several months later, extrapyramidal symptoms increased. On the day of admission to the hospital, the neurologic examination showed abnormalities of upper and lower limbs movements (propulsive walking and the right lower leg traction), the right hand tremor, pouts of the face, and sleepiness.

Key words: extrapyramidal, symptoms, spirochaeta, tick

INTRODUCTION

The disease of Lyme is a tick-bound infectious illness. The skin, the nervous system, joints and the heart are involved. *Spirochaeta (Borrelia burgdorferi)*, which is transmitted by ticks to humans and animals, is the etiologic agent of the disease [1,2]. The infection occurs in spring, summer and autumn. It is passed on to humans by tick sputum, during skin penetration in each generic phase. *Borrelia burgdorferi* invasion needs at least the 24-hour contact with a tick, and the infection risk develops with the contact duration reaching its peak, almost hundred per cent infection risk, on the third day [2].

The clinical symptoms of borreliosis are very diverse and depend on the stage of the disease and organ-related lesions. In the majority of cases, migrating erythema which appears

during 3–30 days is the clinical manifestation of the infection, and sometimes it can occur even to 3 months after a bite from a tick. The classification by Asbrink and Hovmark for the early stage (local or disseminated infection) and the late stage (chronic infection) is commonly accepted. The early phase of infection includes: multiple migrating erythema, early borreliosis, arthritis, myocarditis and other organs lesions. The late phase of chronic infection includes: chronic atrophic skin inflammation, neurologic and rheumatologic symptoms, other organs lesions persisting longer than 12 months.

During this period, durable injuries of affected organs occur [3–5]. Diagnosis of the disease of Lyme is based upon clinical symptoms and serologic tests. The two-step diagnostic protocol is optimal: if the ELISA method is positive, the Western-blot confirming test ought to be performed [6,7]. The biomedica ELISA test is negative <9 BBU/ml (Biomedica *Borrelia* Unit/ml), uncertain 9–11 BBU/ml, positive >11 BBU/ml. During the examination of cerebrospinal fluid the test is positive when the values are >5 BBU/ml. Diagnosis of neuroborreliosis is difficult. Disseminated infection with cerebrospinal abnormalities ought to be confirmed by the medical history of a patient and a positive ELISA test with anti-*Borrelia burgdorferi* serum antibodies, confirmed by the Western-blot test. The cerebrospinal examination confirms the diagnosis, if it

Correspondence to:

Biesiada Grażyna, MD, PhD, Klinika Chorób Zakaźnych, Katedra Gastroenterologii, Hepatologii i Chorób Zakaźnych, Collegium Medicum Uniwersytetu Jagiellońskiego, ul. Śniadeckich 5, 31-501 Kraków, Poland, phone: +48-12-424-73-49, fax: +48-12-424-73-80, e-mail: gbiesiada@op.pl

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shows elevated cells count per milliliter (lymphocytes, monocytes, plasmatic cells and granulocytes), a slightly increased protein level, the presence of specific body-fluids of the immunoglobulin G (IgG) class, and immunoglobulin M (IgM) antibodies confirmed by the ELISA test [8].

The present article describes the case of chronic neuroborreliosis with extrapyramidal symptoms which intensity decreased significantly after the proper antimicrobial medication.

CASE REPORT

A 59-year-old man, a computer specialist, who spent his free time walking in woods for recreation, was admitted to the hospital (history number: 1602/12/04) because of suspected neuroborreliosis. His medical history revealed that 3 years earlier he had been bitten by a tick with following migrating erythema that subsided without antimicrobial treatment. In spite of this, the patient had not changed his hobby exposing himself to bites from ticks. One year later, multiple migrating erythema with following extrapyramidal symptoms, without any organ lesions, occurred. The man was admitted to the Infectious Department and he underwent antimicrobial therapy (ceftriaxon) with neurological improvement. Several months later, increased extrapyramidal symptoms repeated. On the day of admission to the hospital, the neurological examination showed severe abnormalities of upper and lower limb movements (propulsive walking and the right lower leg traction), the right hand tremor, pouts of the face, and sleepiness. The cerebrospinal examination showed cytosis – 2 cells/ μ l, and the cytomorphology revealed a slight inflammatory reaction; protein level was 0.67 g/l, glucose 3.04 mmol/l, chloride 126 mmol/l. Serological tests detecting borreliosis showed: anti-*Borrelia burgdorferi* IgG antibodies, serum level 120 BBU/ml, IgM: 2.9 BBU/ml. The Western-blot test for IgG antibodies indicated 27 points. The magnetic resonance (MR) of the head showed single subcortical demyelination areas placed in both hemispheres. Anti-IgG antibodies level in cerebrospinal fluid was 53.6 BBU/ml, which confirmed their intrameningeal synthesis. Serum serological tests for tick-borne cerebrospinal meningitis detection were negative.

Ceftriaxon in a dose of 2 g once a day, intravenously, was used during 4 weeks. Significant neurological improvement was observed. The control neurological examination showed only a slight tremor of the right hand, the propulsion and traction of the right lower leg were not present. Cytosis 2 cells/ μ l, a small number of macrophages, protein level 0.27 g/l, glucose level 3–4 mmol/l, and chloride 108 mmol/l were observed during the control lumbar puncture. Anti-IgG antibodies level in cerebrospinal fluid was 48.3 BBU/ml. The patient was discharged from the hospital when his clinical condition improved and he was able to walk on his own. Up to the present-day the patient is under the care of the out-

patient clinic. The physical examination performed 6 months later showed further neurological improvement with the persistence of face pouts and a slight tremor of the right hand. Serologic tests showed IgG serum level 59.5 BBU/ml, IgM serum level 6.0 BBU/ml. One year later, the control examination revealed a slight, mainly resting, tremor of the right hand without muscles hyperreflexion, and a less number of synkinetic movements of the right hand. After one month, the patient was admitted to the hospital because of the intensified tremor of the right hand, difficulties in walking, a feeling of cold in limbs, and general weakness. The increased titer of anti-*Borrelia burgdorferi* IgG antibodies to 84.1 BBU/ml was observed. The MR examination of the head showed no progression of the demyelination process in the central nervous system. Antimicrobial treatment was repeated and the following one-year assessment showed the improvement of clinical condition and neurologic status.

DISCUSSION

Suspected neuroborreliosis is connected with particular diagnostic difficulties. The clinical manifestation of neuroborreliosis infection includes: cerebromeningitis, inflammation of cranial nerves, radicular signs, cerebral and spinal cord inflammation and peripheral neuropathy. Clinical symptoms of cerebrospinal inflammation caused by *Borrelia burgdorferi* may be similar to the first episode of multiple sclerosis (sclerosis multiplex). Thorough differential diagnosis is necessary to perform in these cases; specific serum and cerebrospinal fluid antibodies measurement, intrameningeal antibodies synthesis confirmation, and detecting the cerebral localization of demyelination areas, are recommended. Demyelination changes in borreliosis are disseminated differently from the demyelination typical of multiple sclerosis.

In the discussed case, extrapyramidal symptoms, which according to available data rarely occur in the borreliosis infection, were observed. Migrating erythema observed in the described case is sufficient to diagnose the illness as the spirochaeta *Borrelia burgdorferi* infection. Early administration of the proper antimicrobial therapy at the stage of skin lesions is usually efficacious, because it prevents the disease dissemination [2,3,5]. In the discussed case, the patient did not contact the doctor at the clinical stage of skin lesions, and he did not receive any medication. Multiple migrating erythema and early symptoms of neuroborreliosis as the signs of disseminated infection occurred. At the early stage of infection the patient did not contact the physician, so he did not undergo the neurological examination. Based on his medical history, it can be supposed that neurological symptoms formed the extrapyramidal syndrome. During that period the patient did not receive any medication. The treatment was started 2 years after the infection occurred and after the six-month duration of neurological symptoms. Diagnosis was based upon the medical history and high-positive level of anti-*Borrelia burgdorferi* an-

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antibodies in serum and cerebrospinal fluid. The intrameningeal synthesis of antibodies in cerebrospinal space was confirmed. It is particularly important, because these antibodies may passively penetrate the blood-cerebral barrier. The proper antimicrobial treatment resulted in the neurological improvement. In the discussed case this improvement persisted for 13 months. The increased IgG antibodies serum level and no progression revealed by the MR examination accompanied the recurrence of the disease. Repeated antimicrobial therapy lead to the regression of symptoms and up to the present-day, the patient feels good. The appropriateness of the administration of subsequent antimicrobial therapy can be questionable, but in the case of the about one-year delayed significant improvement, antibiotic therapy is recommended.

Early and accurate diagnosis and proper antimicrobial therapy are essential for the treatment of borreliosis. The mere fact of being bitten by a tick is not a sufficient risk agent of the borreliosis infection and does not indicate that all diagnostic and therapeutic procedures should be performed. A patient should be informed about possible pathological symptoms, especially concerning the skin, and after the appearance of any symptoms, he should contact the doctor.

Prophylaxis of borreliosis includes: wearing clothes that protect the body against tick bites, using insect repellents and removing ticks as soon as possible. Pharmacological prophylaxis is recommended when a person who arrived from a non-endemic area to an endemic one suffered multiple bites from ticks.

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